

RBBP8 / CTIP Antibody
Rabbit Polyclonal Antibody
Catalog # ALS13649**Specification**

RBBP8 / CTIP Antibody - Product Information

Application	WB, IHC
Primary Accession	Q99708
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Calculated MW	102kDa KDa

RBBP8 / CTIP Antibody - Additional Information**Gene ID** 5932**Other Names**

DNA endonuclease RBBP8, 3.1.-., CtBP-interacting protein, CtIP, Retinoblastoma-binding protein 8, RBBP-8, Retinoblastoma-interacting protein and myosin-like, RIM, Sporulation in the absence of SPO11 protein 2 homolog, SAE2, RBBP8, CTIP

Target/Specificity

Human CtIP.

Reconstitution & Storage

Aliquot and store at -20°C. Minimize freezing and thawing.

Precautions

RBBP8 / CTIP Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

RBBP8 / CTIP Antibody - Protein Information**Name** RBBP8**Synonyms** CTIP**Function**

Endonuclease that cooperates with the MRE11-RAD50-NBN (MRN) complex in DNA-end resection, the first step of double-strand break (DSB) repair through the homologous recombination (HR) pathway (PubMed:17965729, PubMed:19202191, PubMed:19759395, PubMed:20064462, PubMed:26721387). HR is restricted to S and G2 phases of the cell cycle and preferentially repairs DSBs resulting from replication fork collapse (PubMed:17965729, PubMed:19202191). Key determinant of DSB repair pathway choice, as it commits cells to HR by preventing classical non-homologous end-joining (NHEJ) (PubMed:19202191). Functions downstream of the MRN complex and ATM, promotes ATR activation and its recruitment to DSBs in the S/G2 phase facilitating the generation of ssDNA (PubMed:16581787, PubMed:17965729, PubMed:19759395, PubMed:20064462). Component of the BRCA1-RBBP8 complex that regulates CHEK1 activation and controls cell cycle G2/M checkpoints on DNA damage (PubMed:15485915, PubMed:16818604). During immunoglobulin heavy chain class-switch recombination, promotes microhomology-mediated alternative end joining (A-NHEJ) and plays an essential role in chromosomal translocations (By similarity). Binds preferentially to DNA Y-junctions and to DNA substrates with blocked ends and promotes intermolecular DNA bridging (PubMed:30601117).

Cellular Location

Nucleus. Chromosome. Note=Associates with sites of DNA damage in S/G2 phase (PubMed:10764811, PubMed:25349192). Ubiquitinated RBBP8 binds to chromatin following DNA damage (PubMed:16818604)

Tissue Location

Expressed in ER-positive breast cancer lines, but tends to be down-regulated ER-negative cells (at protein level)

Volume

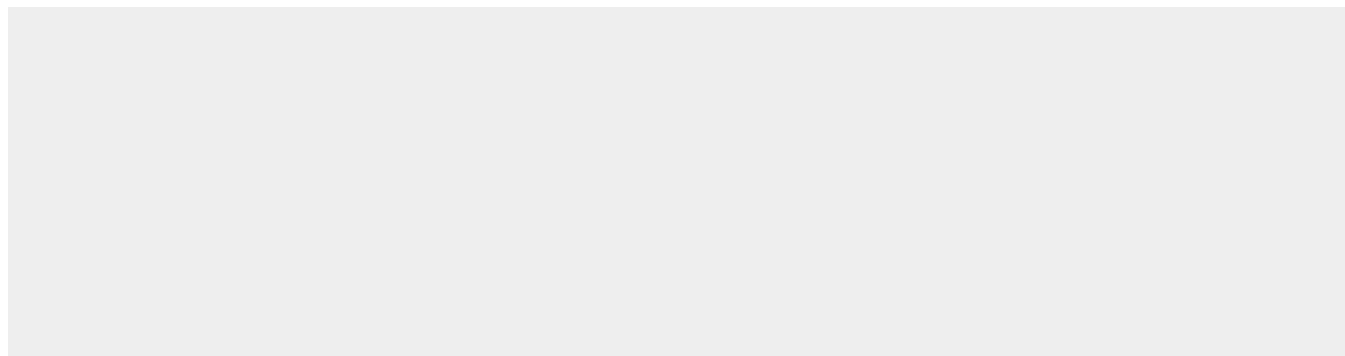
50 µl

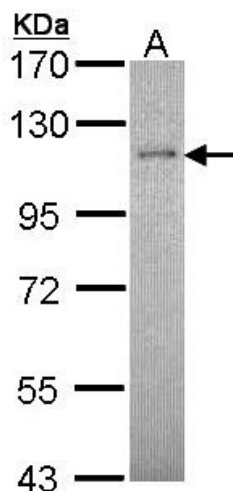
RBBP8 / CTIP Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

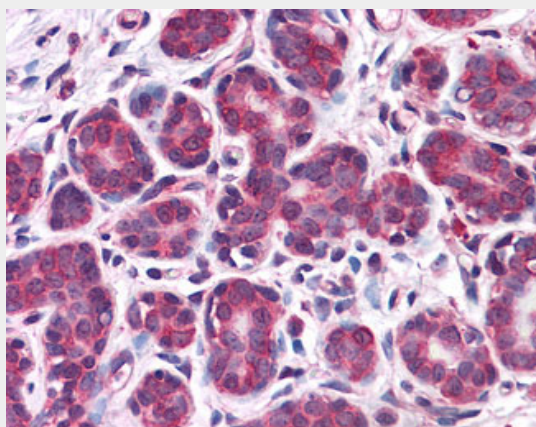
- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

RBBP8 / CTIP Antibody - Images

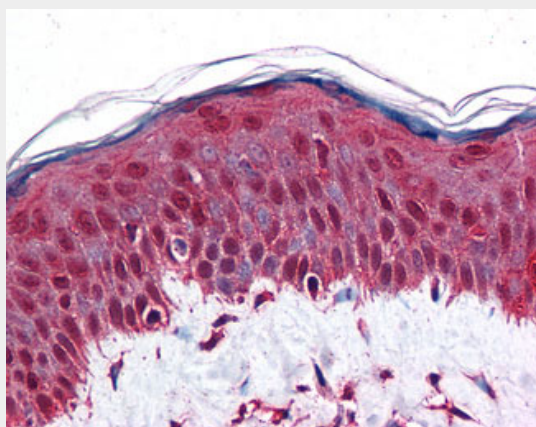




Sample (30 ug of whole cell lysate). A: Hep G2. 7.5% SDS PAGE. RBBP8 antibody diluted at 1:5000.



Anti-RBBP8 / CTIP antibody IHC of human breast.



Anti-RBBP8 / CTIP antibody IHC of human skin.

RBBP8 / CTIP Antibody - Background

Endonuclease that cooperates with the MRE11-RAD50-NBN (MRN) complex in processing meiotic and mitotic double-strand breaks (DSBs) by ensuring both resection and intrachromosomal association of the broken ends. Functions downstream of the MRN complex and ATM, promotes ATR activation and its recruitment to DSBs in the S/G2 phase facilitating the generation of ssDNA.

Component of the BRCA1-RBBP8 complex that regulates CHEK1 activation and controls cell cycle G2/M checkpoints on DNA damage. Promotes microhomology-mediated alternative end joining (A-NHEJ) during class-switch recombination and plays an essential role in chromosomal translocations.

RBBP8 / CTIP Antibody - References

Fusco C., et al. Genomics 51:351-358(1998).
Schaeper U., et al. J. Biol. Chem. 273:8549-8552(1998).
Ota T., et al. Nat. Genet. 36:40-45(2004).
Bechtel S., et al. BMC Genomics 8:399-399(2007).
Nusbaum C., et al. Nature 437:551-555(2005).